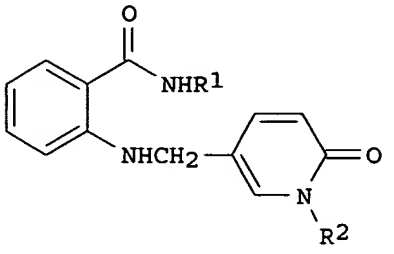
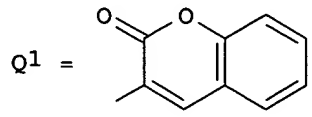
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Dec 2003
Last Updated on STN: 31 Dec 2003

L4 ANSWER 12 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Preparation of N-(pyridinylmethyl)anthranilamides as VEGFR-2 and VEGFR-3 inhibitors for treating diseases caused by persistent angiogenesis
GI



I



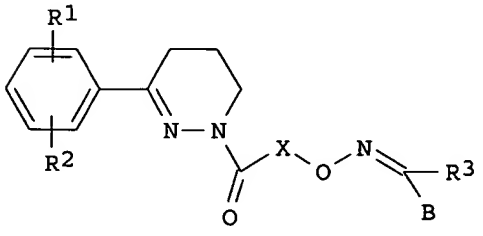
AB Title compds. [I; R1 = (substituted) indazolyl, indolinyl, quinolinyl, Q1; R2 = H, C1-3 alkyl], were prepared Thus, 2-amino-N-(2-oxo-2,3-dihydro-1N-indol-6-yl)benzamide and pyridin-2-one-5-carboxaldehyde in MeOH was treated with ice AcOH followed by stirring over night at room temperature to give 82% N-(2-oxo-2,3-dihydro-1H-indol-6-yl)-2-[(6-oxo-1,6-dihydropyridin-3-yl)methylamino]benzamide. The latter inhibited VEGFR-2 (KDR) with IC50 = 0,05 μM.

ACCESSION NUMBER: 2004:36626 HCAPLUS
TITLE: Preparation of N-(pyridinylmethyl)anthranilamides as VEGFR-2 and VEGFR-3 inhibitors for treating diseases caused by persistent angiogenesis
INVENTOR(S): Huth, Andreas; Krueger, Martin; Zorn, Ludwig; Ince, Stuart; Thierauch, Karl-Heinz; Menrad, Andreas; Haberey, Martin; Hess-Stumpp, Holger
PATENT ASSIGNEE(S): Schering AG, Germany
SOURCE: Ger. Offen., 18 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10228090	A1	20040115	DE 2002-10228090	20020619

PRIORITY APPLN. INFO.: DE 2002-10228090 20020619

L4 ANSWER 13 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Preparation of pyridazinyloximes as phosphodiesterase IV inhibitors.
GI



I

AB Title compds. [I; R1, R2 = H, OH, OR8, SR8, SOR8, SO2R8, halo; R1R2 = OCH2O, OCH2CH2O; R3 = H, AR7, COAR7, CO2AR7, CONH2, NH2, etc.; R7 = H, CO2H, NH2, OH, etc.; R8 = (substituted) alkyl, alkenyl, cycloalkyl, alkylenecycloalkyl, etc.; A = null, (O, S, SO, SO2, imino-interrupted) alkylene, alkenylene, cycloalkylene; B = (substituted) aryl, heteroaryl; X = (O, S, SO, SO2, imino-interrupted) alkylene], were prepared as phosphodiesterase IV inhibitors for treating osteoporosis, tumors, cachexia, atherosclerosis, rheumatoid arthritis, multiple sclerosis, diabetes mellitus, inflammatory processes, allergies, asthma, autoimmune diseases, myocardial diseases and AIDS (no data). Thus, 3-(3-ethoxy-4-methoxyphenyl)-5,6-dihydro-4H-pyridazine was treated sequentially with chloroacetyl chloride, N-hydroxyphthalimide, ethanolamine, and 4-methoxybenzaldehyde to give 4-methoxybenzaldehyde O-[2-[3-(3-ethoxy-4-methoxyphenyl)-5,6-dihydro-4H-pyridazin-1-yl]-2-oxoethyl]oxime.

ACCESSION NUMBER: 2003:991488 HCAPLUS
DOCUMENT NUMBER: 140:27834
TITLE: Preparation of pyridazinyloximes as phosphodiesterase IV inhibitors.
INVENTOR(S): Eggenweiler, Hans-Michael; Beier, Norbert; Schelling, Pierre; Wolf, Michael
PATENT ASSIGNEE(S): Merck Patent G.m.b.H., Germany
SOURCE: PCT Int. Appl., 137 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003104205	A1	20031218	WO 2003-EP5173	20030516
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 10225574	A1	20031218	DE 2002-10225574	20020610

PRIORITY APPLN. INFO.: DE 2002-10225574 A 20020610
OTHER SOURCE(S): MARPAT 140:27834
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Basic non-peptide bradykinin antagonists, particularly 3-(8-quinolinoxymethyl)benzenesulfonamide derivatives of α,α-dialkyl amino acids, with specific B2 receptor antagonist activity, and pharmaceutical compositions therefrom
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Non-peptide compds. of formula I, having activity as specific antagonists of bradykinin (BK) B2 receptor, are disclosed [wherein: R1 = H or C1-4

alkyl; R2, R3 = C1-4 alkyl; or R2 and R3 form a 3- to 7-membered (hetero)cyclic aliphatic group with 0-2 N/O/S atoms; R4, R5 = H, C1-4 alkyl; X = halo, OR1, SR1, CN, or C1-4 alkyl; B = variety of groups with at least 1 amino group of basic character or a tetraalkylammonium group, typically with 1 or 2 such groups, selected from particular cyclic and acyclic structures; including particular pharmacol. acceptable salts with (in)organic acids, and including optical isomers and their (non)racemic mixts.]. Compds. I are chemical characterized by the presence of an alpha,alpha-disubstituted amino acid residue, and at least one addnl. amino group, free or salified, or the corresponding ammonium quaternary salt. I are a novel class of medicaments, which can be used in treating a variety of disorders in which B2 receptors are involved. Approx. 90 example compds. and approx. 20 intermediates are described. For instance, invention compound II was prepared as the trifluoroacetate salt in 26% yield by EDC coupling of a Boc-protected aminohexanoic acid derivative with the corresponding piperazine derivative, followed by deprotection. In a test for binding to human B2 receptor expressed in human fibroblasts W138, invention compound III had a pKi of 10.1. Compds. I also inhibited bradykinin-induced bronchospasm in guinea pigs (no data), showing a higher potency and longer duration than similar mols. not containing the α,α-dialkyl amino acid moiety.

ACCESSION NUMBER: 2003:991349 HCAPLUS
DOCUMENT NUMBER: 140:42038
TITLE: Basic non-peptide bradykinin antagonists, particularly 3-(8-quinolinoxymethyl)benzenesulfonamide derivatives of α,α-dialkyl amino acids, with specific B2 receptor antagonist activity, and pharmaceutical compositions therefrom
INVENTOR(S): Calvani, Frederico; Catrambone, Fernando; Felicetti, Patrizia; Fincham, Christopher Ingo; Giolitti, Alessandro; Maggi, Carlo Alberto; Quartara, Laura; Rossi, Cristina; Terracciano, Rosa
PATENT ASSIGNEE(S): Menarini Ricerche S.P.A., Italy
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003103671	A1	20031218	WO 2003-EP5893	20030605

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: IT 2002-MI1247 A 20020607
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 15 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Modified anti-tumor necrosis factor immunoglobulins containing extra constant region Ig domain inserted into its constant region and their therapeutic uses
AB The present invention relates to modified anti-tumor necrosis factor Igs. The modified anti-TNF Igs contains an extra constant region Ig domain

SOURCE: PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003093787	A2	20031113	WO 2003-US13154	20030428

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2002-376996P P 20020430

L4 ANSWER 19 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Preparation of 2-phenyl-3(2H)-pyridazinones as lysyl oxidase inhibitors for preventing and treating **fibrosis**
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Title compds. I [wherein R1 = (un)substituted 5- to 7-membered heterocyclyl ring selected from imidazolyl, triazolyl, pyridinyl, piperazinyl, 1,4-diazacycloheptyl, morpholinyl, thiomorpholinyl, etc.; R2 = (un)substituted (hetero)aryl; R3 = H, halo, alkyl, CF3, NO2, CN, CO2H or alkoxycarbonyl; and their salts, solvates, and solvates of their salts] were prepared as lysyl oxidase inhibitors for preventing and treating **fibrosis** in humans and/or animals. For example, II was prepared by alkylation of tert-Bu 1-piperazinecarboxylate with 2-(4-chlorophenyl)-4,5-dichloro-3(2H)-pyridazinone in dioxane in the presence of NaI at 100°, reaction of the 5-chloropyridazinone intermediate with potassium 4-phenylphenoxide in DMF, followed by Boc-deprotection. Selected I exhibited excellent IC50 values in the range of 0.003 μM to 0.017 μM for the inhibition of lysyl oxidase compared to BAPN (10 μM) and structurally related emorfazone (> 4 μM). Selected I were tested for their antifibrotic activity in rats and were found active in the chronic CCl4 poisoning model, the bile duct ligature model, and the serum-induced liver **fibrosis** model.

ACCESSION NUMBER: 2003:872263 HCAPLUS
DOCUMENT NUMBER: 139:364943
TITLE: Preparation of 2-phenyl-3(2H)-pyridazinones as lysyl oxidase inhibitors for preventing and treating **fibrosis**
INVENTOR(S): Schohe-Loop, Rudolf; Burchardt, Elmar; Faeste, Christiane; Hirth-Dietrich, Claudia; Keldenich, Joerg; Knorr, Andreas; Lampe, Thomas; Naab, Paul; Schmidt, Delf; Schmidt, Gunther
PATENT ASSIGNEE(S): Bayer AG, Germany
SOURCE: Ger. Offen., 106 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10216144	A1	20031106	DE 2002-10216144	20020412
WO 2003097612	A1	20031127	WO 2003-EP3628	20030408

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DE 2002-10216144 A 20020412
OTHER SOURCE(S): MARPAT 139:364943

L4 ANSWER 20 OF 95 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Treatment of osteoarthritis
AB Agents with integrin-affecting activity, including antibodies and mols. having the antigen-binding portion of such antibodies, are used to regulate inflammatory mediators, including IL-1β, IL-6, IL-8, nitric oxide, PGE2 and MMPs.
ACCESSION NUMBER: 2003:855390 HCAPLUS
DOCUMENT NUMBER: 139:317448
TITLE: Treatment of osteoarthritis
INVENTOR(S): Amin, Ashok R.; Abramson, Steven; Attur, Mukandan
PATENT ASSIGNEE(S): New York University, USA
SOURCE: U.S. Pat. Appl. Publ., 30 pp., Cont.-in-part of U.S. Ser. No. 441,217.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003202977	A1	20031030	US 2003-461423	20030616

PRIORITY APPLN. INFO.: US 1998-108521P P 19981116
US 1999-116966P P 19990122
US 1999-441217 B1 19991116

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(FILE 'HOME' ENTERED AT 16:15:06 ON 04 FEB 2004)

FILE 'STNGUIDE' ENTERED AT 16:15:13 ON 04 FEB 2004

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JAPIO, BIOSIS, CEN, CEABA-VTB, BIOBUSINESS, HCAPLUS' ENTERED AT 16:16:22 ON 04 FEB 2004

L1 993 S FIBROSIS () TREATMENT
L2 45818 S FIBROSIS AND CIRRHOSIS
L3 1 S CHRONIC PANCREATITUS
L4 95 S L2 AND L1
L5 2524 S F-MET-LEU
L6 346 S N-FORMYL PEPTIDES
L7 0 S L6 AND L1
L8 1 S L5 AND L1

=> s l5 and l6
L9 19 L5 AND L6

=> d l9 ti abs ibib tot

L9 ANSWER 1 OF 19 MEDLINE on STN

TI In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: flow cytometric evidence for the selective absence of formyl peptide receptors.

AB Interest in the role that activated granulocytes play in C5a-induced myocardial ischemia prompted us to investigate and compare activation responses of pig and human neutrophils. The responses of Hypaque-Ficoll purified porcine (P-PMN) and human neutrophils (H-PMN) to stimulation with N-formyl-methionyl-leucyl-phenylalanine (FMLP), C5a, phorbol myristate acetate (PMA), and calcium ionophore A23187 (A23187) were compared by flow cytometrically measured changes in the cells' forward (FWD-SC) (a measure of shape/volume change) and right angle (90 degrees-SC) light scatter (a measure of secretion), and in the distribution of the membrane potential sensitive fluorescent probe di-O-C (3). FMLP, C5a, and Zymosan-activated serum (ZAS stimulated chemotaxis and FMLP vs. PMA-stimulated adherence to plastic were also compared. Unstimulated P-PMN had lower FWD-SC and 90 degrees-SC than H-PMN (39.4 +/- 1.4 vs. 48.4 +/- 2.0 P less than 0.05, and 32.7 +/- 2.7 vs. 52.4 +/- 1.5 units, P less than 0.005, for FWD-SC and 90 degrees-SC of P-PMN vs. H-PMN, respectively). P-PMN selectively failed to increase their FWD-SC upon stimulation with FMLP (0.0 +/- 0.5% vs. 26.1 +/- 6.8%, P-PMN vs. H-PMN), or decrease their 90 degrees-SC when treated with cytochalasin B + FMLP (secretion) (2.4 +/- 0.1% vs. -35.8 +/- 4.6% change in 90 degrees-SC, P-PMN vs. H-PMN), while responding comparably to C5a, PMA, and A23187. P-PMN failed to depolarize in response to FMLP but responded similarly to H-PMN when activated by C5a, A23187, and PMA. P-PMN's chemotactic response to FMLP was selectively absent since the cells responded well to purified pig C5a. FMLP stimulated significant increases in H-PMN adherence to bovine serum albumin-coated plastic (44.1 +/- 6.7% vs. 12.6 +/- 3.7%, FMLP vs. buffer, P less than 0.025), but failed to increase adherence of P-PMN above baseline 0.68 +/- 0.20% vs. 2.12 +/- 1.90%, FMLP vs. buffer, P greater than 0.05. PMA (100 ng/ml) stimulated comparable increases in adherence in both PMN types (48.6 +/- 5.2% vs. 58.7 +/- 4.9%, P-PMN vs. H-PMN, P less than 0.025). Binding studies using the fluoresceinated N-formyl peptide **f-met**-**leu**-phe-lysine-fluorescein-isothiocyanate (FMLPL-FITC) in the absence and presence of excess non-fluoresceinated FMLPL indicated that P-PMN lack specific binding sites for the **N-formyl peptides**.(ABSTRACT TRUNCATED AT 400 WORDS)

ACCESSION NUMBER: 90203813 MEDLINE
DOCUMENT NUMBER: 90203813 PubMed ID: 2108228
TITLE: In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: flow cytometric evidence for the selective absence of formyl peptide receptors.
AUTHOR: Fletcher M P; Stahl G L; Longhurst J C
CORPORATE SOURCE: Division of Rheumatology/Allergy and Clinical Immunology, School of Medicine, University of California, Davis 95616.
CONTRACT NUMBER: P30-AM 35747-01 (NIADDK)
SOURCE: JOURNAL OF LEUKOCYTE BIOLOGY, (1990 Apr) 47 (4) 355-65. Journal code: 8405628. ISSN: 0741-5400.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900504

L9 ANSWER 2 OF 19 MEDLINE on STN

TI Evidence for the presence of specific receptors for N-formyl chemotactic peptides on human spermatozoa.

AB Synthetic N-formylated peptides are potent chemoattractants for human spermatozoa in vitro. The specific structure-activity relations for eliciting a chemotactic response and the ability of the antagonist tertbutoxycarbonyl-phenylalanyl-leucyl-phenylalanyl-leucyl- phenylalanine (Boc-Phe-Leu-Phe-Leu-Phe) to inhibit the chemotaxis induced by these peptides strongly suggest the presence of receptors on human spermatozoa. The following studies were performed to identify specific binding sites on human spermatozoa by using [35S]-N-formyl-methionyl-leucyl-phenylalanine [(35S]**f-Met-Leu**-Phe), a potent chemotactic peptide. Binding of the [35S]formyl-peptide to human spermatozoa was rapid (t1/2, 8 min) and reversible. Binding isotherms of the saturation experiments revealed a single class of high affinity, low capacity binding sites (equilibrium dissociation constant, 17.7 nM; maximal binding, 109 fmol/2 X 10(6) cells) and an average number of 60,000 receptors per cells. The biological potencies of a series of formyl peptides as chemoattractants correlated closely with their relative abilities to compete with [35S]**f-Met-Leu**-Phe for specific binding to human spermatozoa. These data fulfill the major criteria for demonstration of specific receptors for chemotactic peptides on human spermatozoa. It is likely that these receptor sites initiate the chemotactic response of human spermatozoa to **N-formyl peptides**.

ACCESSION NUMBER: 86304837 MEDLINE
DOCUMENT NUMBER: 86304837 PubMed ID: 3018025
TITLE: Evidence for the presence of specific receptors for N-formyl chemotactic peptides on human spermatozoa.
AUTHOR: Gnessi L; Fabbri A; Silvestroni L; Moretti C; Fraioli F; Pert C B; Isidori A
SOURCE: JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1986 Oct) 63 (4) 841-6.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198610
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19861015

L9 ANSWER 3 OF 19 USPATFULL on STN

TI Biochips and method of screening using drug induced gene and protein expression profiling

AB The present invention provides relates to a biochip microarray, with multiple properties for use in identification of gene- and protein-induction or repression by drugs, the evaluation of efficacy and toxicity of any drug of choice, prediction of efficacy and toxicity of newly-discovered drugs, families of drugs or classes of drugs. Experimental information acquired from the biochip is inputted into a Drug-Gene-Protein-Biology (DGPB) database from which experimental data can be mined and analyzed based on the users preferences. A method for predicting the effect of a test composition for the treatment of a disease also is described. An animal model for the diseases selected. A biochip array for evaluating the effect of the test composition for the treatment of the disease is provided. The test composition is tested in the animal model to obtain a first set of biological markers representative of the effect of the test composition in the animal model. The biochip array generates a first set of data representative of the first set of biological markers. The first set of data is evaluated to predict the effect of the test composition on the disease.

Preferably, the animal model is a standard animal model for human disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:180722 USPATFULL
TITLE:	Biochips and method of screening using drug induced gene and protein expression profiling
INVENTOR(S):	Lindemann, Garrett W., Benicia, CA, UNITED STATES Lipani, John, Fountain Hills, AZ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003124552	A1	20030703	
APPLICATION INFO.:	US 2002-140680	A1	20020508	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-289407P	20010508	(60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	EDWARDS & ANGELL, LLP, P.O. BOX 9169, BOSTON, MA, 02209
NUMBER OF CLAIMS:	23
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	2 Drawing Page(s)
LINE COUNT:	3520

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 19 USPATFULL on STN

TI Methods and compositions for identifying receptor effectors

AB The present invention makes available a rapid, effective assay for screening and identifying pharmaceutically effective compounds that specifically interact with and modulate the activity of a cellular receptor or ion channel. The subject assay enables rapid screening of large numbers of polypeptides in a library to identifying those polypeptides which induce or antagonize receptor bioactivity. The subject assay is particularly amenable for identifying surrogate ligands for orphan receptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:78482 USPATFULL
TITLE:	Methods and compositions for identifying receptor effectors
INVENTOR(S):	Klein, Christine A., Ossining, NY, UNITED STATES Murphy, Andrew J.M., Croton on the Hudson, NY, UNITED STATES Fowlkes, Dana M., Chapel Hill, NC, UNITED STATES Broach, James, Princeton, NJ, UNITED STATES Manfredi, John, Ossining, NY, UNITED STATES Paul, Jeremy, Nyack, NY, UNITED STATES Trueheart, Joshua, Nyack, NY, UNITED STATES
PATENT ASSIGNEE(S):	Cadus Pharmaceutical Corporation (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003054402	A1	20030320	
APPLICATION INFO.:	US 2001-953354	A1	20010913	(9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1996-689172, filed on 6 Aug 1996, ABANDONED Continuation-in-part of Ser. No. US 1996-582333, filed on 17 Jan 1996, GRANTED, Pat. No. US 6255059 Continuation-in-part of Ser. No. US 1994-322137, filed on 13 Oct 1994, GRANTED, Pat. No. US 6100042 Continuation-in-part of Ser. No. US 1994-309313, filed on 20 Sep 1994, ABANDONED Continuation-in-part of Ser. No. US 1994-190328, filed			

on 31 Jan 1994, ABANDONED Continuation-in-part of Ser. No. US 1993-41431, filed on 31 Mar 1993, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 76
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 5008
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 19 USPATFULL on STN
TI Complexes of alpha-6 integrin subunits with small peptides and methods for treating indications resulting from modulation of integrin-mediated responses by altering signal transduction
AB A method for modulating an alpha 6 subunit containing integrin-mediated signal transduction is described. The method involves contacting a cell with an effective integrin modulating amount of an alpha 6 subunit containing integrin-mediated signal transduction pathway modification agent. Preferred agents are peptides having the formula **f-Met-Leu-X**, wherein X is selected from the group consisting of Tyr, Tyr-Phe, Phe-Phe and Phe-Tyr.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:71962 USPATFULL
TITLE: Complexes of alpha-6 integrin subunits with small peptides and methods for treating indications resulting from modulation of integrin-mediated responses by altering signal transduction
INVENTOR(S): Clagett, James A., Snohomish, WA, UNITED STATES
Lipani, John, Mountain Hills, AZ, UNITED STATES
Palmer, Craig Robert, San Francisco, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003050249	A1	20030313	
APPLICATION INFO.:	US 2001-863837	A1	20010523	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-206397P	20000523 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dike, Bronstein, Roberts & Cushman, Intellectual Property Practice Group, Edwards & Angell, LLP, 101 Federal Street, Boston, MA, 02209
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 1457
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 19 USPATFULL on STN
TI Methods and compositions for identifying receptor effectors
AB The present invention makes available a rapid, effective assay for screening and identifying pharmaceutically effective compounds that specifically interact with and modulate the activity of a cellular receptor or ion channel. The subject assay enables rapid screening of large numbers of polypeptides in a library to identifying those polypeptides which induce or antagonize receptor bioactivity. The subject assay is particularly amenable for identifying surrogate ligands for orphan receptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2001:170878 USPATFULL
TITLE: Methods and compositions for identifying receptor effectors
INVENTOR(S): Klein, Christine A., Ossining, NY, United States
Murphy, Andrew J., Croton-on-Hudson, NY, United States
Fowlkes, Dana M., Chapel Hill, NC, United States
Broach, James, Princeton, NJ, United States
Manfredi, John, Ossining, NY, United States
Paul, Jeremy, Nyack, NY, United States
Trueheart, Joshua, South Nyack, NY, United States
PATENT ASSIGNEE(S): Cadus Pharmaceutical Corporation. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001026926	A1	20011004
APPLICATION INFO.:	US 2000-747774	A1	20001221 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1996-582333, filed on 17 Jan 1996, GRANTED, Pat. No. US 6255059 Continuation-in-part of Ser. No. US 1995-464531, filed on 5 Jun 1995, GRANTED, Pat. No. US 5789184 Continuation-in-part of Ser. No. US 1995-461598, filed on 5 Jun 1995, GRANTED, Pat. No. US 5876951 Continuation-in-part of Ser. No. US 1995-461383, filed on 5 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1995-463181, filed on 5 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1994-322137, filed on 13 Oct 1994, GRANTED, Pat. No. US 6100042 Continuation-in-part of Ser. No. US 1994-309313, filed on 20 Sep 1994, ABANDONED Continuation-in-part of Ser. No. US 1994-190328, filed on 31 Jan 1994, ABANDONED Continuation-in-part of Ser. No. US 1993-41431, filed on 31 Mar 1993, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 76
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 4641
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 19 USPATFULL on STN
TI Methods for identifying G protein coupled receptor effectors
AB The present invention makes available a rapid, effective assay for screening and identifying pharmaceutically effective compounds that specifically interact with and modulate the activity of a cellular receptor or ion channel. The subject assay enables rapid screening of large numbers of polypeptides in a yeast expression library to identifying those polypeptides which induce or antagonize receptor bioactivity. The subject assay is particularly amenable for identifying surrogate ligands for orphan receptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2001:102569 USPATFULL
TITLE: Methods for identifying G protein coupled receptor effectors
INVENTOR(S): Klein, Christine A., Ossining, NY, United States
Murphy, Andrew J. M., Montclair, NJ, United States
Fowlkes, Dana M., Chapel Hill, NC, United States
Broach, James, Princeton, NJ, United States
Manfredi, John, Ossining, NY, United States
Paul, Jeremy, Nyack, NY, United States
Trueheart, Joshua, South Nyack, NY, United States
PATENT ASSIGNEE(S): Cadus Pharmaceutical Corporation, Tarrytown, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6255059	B1	20010703
APPLICATION INFO.:	US 1996-582333		19960117 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-463181, filed on 5 Jun 1995, now abandoned Continuation-in-part of Ser. No. US 1994-322137, filed on 13 Oct 1994 Continuation-in-part of Ser. No. US 1994-309313, filed on 20 Sep 1994, now abandoned Continuation-in-part of Ser. No. US 1994-190328, filed on 31 Jan 1994, now abandoned Continuation-in-part of Ser. No. US 1993-41431, filed on 31 Mar 1993, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Spector, Lorraine
ASSISTANT EXAMINER: Kaufman, Claire M.
LEGAL REPRESENTATIVE: Lahive & Cockfield, LLP, DeConti, Jr., P, Giulio A., Lauro, Esq., Peter C.
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 4507
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 19 USPATFULL on STN
TI Recombinant yeast cells for identifying receptor effectors
AB The present invention makes available a rapid, effective assay for screening and identifying pharmaceutically effective compounds that specifically interact with and modulate the activity of a cellular protein, e.g., a receptor or ion channel. The subject assay enables rapid screening of large numbers of compounds to identify those which act as an agonist or antagonist to the bioactivity of the cellular protein. The subject assay is particularly amenable for identifying surrogate ligands for receptors especially from small molecule or peptide libraries or from peptides produced by an autocrine system.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:167753 USPATFULL
TITLE: Recombinant yeast cells for identifying receptor effectors
INVENTOR(S): Trueheart, Joshua, Concord, MA, United States
Paul, Jeremy I., Nyack, NY, United States
Fuernkranz, Hans A., San Jose, CA, United States
Nathan, Debra, Mt. Kisco, NY, United States
Holmes, Scott, Middlebury, CT, United States
PATENT ASSIGNEE(S): Cadus Pharmaceutical Corporation, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6159705		20001212
APPLICATION INFO.:	US 1997-936632		19970924 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-718910, filed on 24 Sep 1996, now abandoned And a continuation-in-part of Ser. No. US 1997-851469, filed on 5 May 1997, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Ulm, John
LEGAL REPRESENTATIVE: Lahive & Cockfield, LLP, DeConti, Jr., Esq., Giulio A., Lauro, Esq., Peter C.
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 5260
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 19 USPATFULL on STN
TI Formyl-methionyl chemotatic peptide antibiotic conjugates useful in treating infections
AB A group of synthetic N-formyl methionine tri and tetra peptides in covalent combination with antibiotics are useful in treating infections. These peptideantibiotic conjugates exhibit a high degree of chemotactic activity for polymorphonuclear leukocytes and monocytes while simultaneously inhibiting the growth of microorganisms. The use of chemotactic peptide-silver sulfadiazine conjugates is particularly effective for treating burns.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 84:4567 USPATFULL
TITLE: Formyl-methionyl chemotatic peptide antibiotic conjugates useful in treating infections
INVENTOR(S): Schiffman, Elliott, Chevy Chase, MD, United States
Altman, Leonard C., Seattle, WA, United States
PATENT ASSIGNEE(S): Research Corporation, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 4427660		19840124	
APPLICATION INFO.:	US 1982-354357		19820303	(6)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Phillips, Delbert R.
LEGAL REPRESENTATIVE: Scully, Scott, Murphy and Presser
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 941
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 19 EMBASE on STN
TI In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: Flow cytometric evidence for the selective absence of formyl peptide receptors.
AB Interest in the role that activated granulocytes play in C5a-induced myocardial ischemia prompted us to investigate and compare activation responses of pig and human neutrophils. The responses of Hypaque-Ficoll purified porcine (P-PMN) and human neutrophils (H-PMN) to stimulation with N-formyl-methionyl-leucyl-phenylalanine (FMLP), C5a, phorbol myristate acetate (PMA), and calcium ionophore A23187 (A23187) were compared by flow cytometrically measured changes in the cells' forward (FWD-SC) (a measure of shape/volume change) and right angle (90°-SC) light scatter (a measure of secretion), and in the distribution of the membrane potential sensitive fluorescent probe di-O-C((5)) (3). FMLP, C5a, and Zymosan-activated serum (ZAS) stimulated chemotaxis and FMLP vs. PMA-stimulated adherence to plastic were also compared. Unstimulated P-PMN had lower FWD-SC and 90°-SC than H-PMN (39.4 ± 1.4 vs. 48.4 ± 2.0 $P < 0.05$, and 32.7 ± 2.7 vs. 52.4 ± 1.5 units, $P < 0.005$, for FWD-SC and 90°-SC of P-PMN vs. H-PMN, respectively). P-PMN selectively failed to increase their FWD-SC upon stimulation with FMLP (0.0 ± 0.5% vs. 26.1 ± 6.8%, P-PMN vs. H-PMN), or decrease their 90°-SC when treated with cytochalasin B + FMLP (secretion) (2.4 ± 0.1% vs. -35.8 ± 4.6% change in 90°-SC, P-PMN vs. H-PMN), while responding comparably to C5a, PMA, and A23187. P-PMN failed C5a, A23187, and PMA. P-PMN's chemotactic response to fMLP was selectively

absent since the cells responded well to purified pig C5a. FMLP stimulated significant increases in H-PMN adherence to bovine serum albumin-coated plastic (44.1 ± 6.7% vs. 12.6 ± 3.7%, FMLP vs. buffer, P < 0.025), but failed to increase adherence of P-PMN above baseline 0.68 ± 0.20% vs. 2.12 ± 1.90%, FMLP vs. buffer, P > 0.05. PMA (100 ng/ml) stimulated comparable increases in adherence in both PMN types (48.6 ± 5.2% vs. 58.7 ± 4.9%, P-PMN vs. H-PMN, P < 0.025). Binding studies using the fluoresceinated N-formyl peptide **f-met-leu**-phe-lysine-fluorescein-isothiocyanate (FMLPL-FITC) in the absence and presence of excess non-fluoresceinated FMLPL indicated that P-PMN lack specific binding sites for the **N-formyl peptides**. Intracoronary (LAD) infusion of FMLP in the instrumented intact pig produced no change in neutrophil extraction, LAD regional blood flow, or myocardial contractility while infusion of purified porcine C5a induced a rapid and marked increase in myocardial neutrophil extraction, a decrease in LAD coronary blood flow, and diminished contractility. It is concluded that P-PMN and H-PMN respond comparably to C5a, PMA, and A23187, but P-PMN are selectively unresponsive to activation by FMLP both in vitro and in vivo due to the absence of FMLP binding.

ACCESSION NUMBER: 90117677 EMBASE
DOCUMENT NUMBER: 1990117677
TITLE: In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: Flow cytometric evidence for the selective absence of formyl peptide receptors.
AUTHOR: Fletcher M.P.; Stahl G.L.; Longhurst J.C.
CORPORATE SOURCE: Div. of Rheumatology/Allergy, School of Medicine, University of California,Davis, CA 95616, United States
SOURCE: Journal of Leukocyte Biology, (1990) 47/4 (355-365). ISSN: 0741-5400 CODEN: JLBIE7
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 11 OF 19 EMBASE on STN

TI Evidence for the presence of specific receptors for N-formyl chemotactic peptides on human spermatozoa.

AB Synthetic N-formylated peptides are potent chemoattractants for human spermatozoa in vitro. The specific structure-activity relations for eliciting a chemotactic response and the ability of the antagonist terbutoxycarbonyl-phenylalanyl-leucyl-phenylalanyl-leucyl-ph enylalanine (Boc-Phe-Leu-Phe-Leu-Phe) to inhibit the chemotaxis induced by these peptides strongly suggest the presence of receptors on human spermatozoa. The following studies were performed to identify specific binding sites on human spermatozoa by using [35S]-N-formyl-methionyl-leucyl-phenilalanine ([35S]**f-Met-Leu**-Phe), a potent chemotactic peptide. Binding of the [35S]formyl-peptide to human spermatozoa was rapid (t1/2, 8 min) and reversible. Binding isotherms of the saturation experiments revealed a single class of high affinity, low capacity binding sites (equilibrium dissociation constant, 17.7 nM; maximal binding, 109 fmol/2 x 106 cells) and an average number of 60,000 receptors per cell. The biological potencies of a series of formyl peptides as chemoattractants correlated closely with their relative abilities to compete with [35S]**f-Met-Leu**-Phe for specific binding to human spermatozoa. These data fulfill the major criteria for demonstration of specific receptors for chemotactic peptides on human spermatozoa. It is likely that these receptor sites initiate the chemotactic response of human spermatozoa to **N-formyl-peptides**.

ACCESSION NUMBER: 86251784 EMBASE
DOCUMENT NUMBER: 1986251784
TITLE: Evidence for the presence of specific receptors for N-formyl chemotactic peptides on human spermatozoa.
AUTHOR: Gnessi L.; Fabbri A.; Silvestroni L.; et al.
CORPORATE SOURCE: V Clinica Medica, Policlinico Umberto I, Universita' La Sapienza, 00161 Rome, Italy
SOURCE: Journal of Clinical Endocrinology and Metabolism, (1986) 63/4 (841-846).
CODEN: JCEMAZ
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 029 Clinical Biochemistry
003 Endocrinology
002 Physiology
LANGUAGE: English

L9 ANSWER 12 OF 19 EMBASE
on STN
TI Modulating influence of chemotactic factor-induced cell adhesiveness on granulocyte function.
AB The importance of adhesion in regulating locomotion and accumulation of polymorphonuclear leukocytes (PMN) has remained vague. We found that the chemotaxis of human PMN resuspended in heat-inactivated plasma was maximal toward 1-10 nM N-formyl-met-leu-phe (**f-Met-Leu**-Phe), but fell below random motility toward ≥100 nM. This impressive decrease of motility was paralleled by increased cell adherence on Petri dishes being minimal at 1 nM and maximal at >10 nM **f-Met-Leu**-Phe (6 ± 1 and 37 ± 2% [SE] adherent cells, respectively). Checked by phase-contrast microscopy, cells under stimulated adhesion lost the typical bipolar shape of moving PMN and became immobilized and highly flattened. PMN, preexposed to 250 nM **f-Met-Leu**-Phe and tested after washing, retained increased adhesiveness and showed extremely low random and chemotactic motility. In contrast, preexposure to 1 nM **f-Met-Leu**-Phe had no effect on chemotaxis. Supporting the concept that immobilizing hyperadhesiveness does not correspond to a general functional hyporesponsiveness of PMN, no depression of the initial ingestion rate was observed in the presence of 250 nM **f-Met-Leu**-Phe. Moreover, a close correlation was found between the induction of PMN adhesiveness and the stimulation of the hexose monophosphate pathway activity as well as of lysomal enzyme release ($r \geq 0.98$). Thus, 'chemotactic deactivation' and 'high-dose inhibition of chemotaxis' by **N-formyl peptides** is the consequence of increased cell adhesiveness. This phenomenon provides a mechanism for cell trapping at the inflammatory site. Conversely, if operative in circulating blood, e.g., in septicemia, it may impair PMN emigration to such sites.
ACCESSION NUMBER: 79217084 EMBASE
DOCUMENT NUMBER: 1979217084
TITLE: Modulating influence of chemotactic factor-induced cell adhesiveness on granulocyte function.
AUTHOR: Fehr J.; Dahinden C.
CORPORATE SOURCE: Dept. Med., Sect. Hematol. CH 5, Univ. CH-8091 Zurich, Switzerland
SOURCE: Journal of Clinical Investigation, (1979) 64/1 (8-16).
CODEN: JCINAO
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
025 Hematology
026 Immunology, Serology and Transplantation
030 Pharmacology
LANGUAGE: English

L9 ANSWER 13 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

TI IN-VIVO AND IN-VITRO ASSESSMENT OF PORCINE NEUTROPHIL ACTIVATION RESPONSES TO CHEMOATTRACTANTS FLOW CYTOMETRIC EVIDENCE FOR THE SELECTIVE ABSENCE OF FORMYL PEPTIDE RECEPTORS.

AB Interest in the role that activated granulocytes play in C5a-induced myocardial ischemia prompted us to investigate and compare activation responses of pig and human neutrophils. The responses of Hypaque-Ficoll purified porcine (P-PMN) and human neutrophils (H-PMN) to stimulation with N-formyl-methionyl-leucyl-phenylalanine (FMLP), C5a,phorbol myristate acetate (PMA), and calcium ionophore A23187 (A23187) were compared by flow cytometrically measured changes in the cells forward (FWD-SC) (a measure of shape/volume change) and right angle (90°-SC) light scatter (a measure of secretion), and in the distribution of the membrane potential sensitive fluorescent probe di-O-C(5) (3). FMLP, C5a, and Zymosan-activated serum (ZAS) stimulated chemotaxis and FMLP vs. PMA-stimulated adherence to plastic were also compared. Unstimulated P-PMN had lower FWD-SC and 90°-SC than H-PMN (39.4 ± 1.4 vs. 48.4 ± 2.0 P < 0.05, and 32.7 ± 2.7 vs. 52.4 ± 1.5 units, P < 0.005, for FWD-SC and 90°-SC of P-PMN vs. H-PMN, respectively). P-PMN selectively failed to increase their FWD-SC upon stimulation with FMLP (0.0 ± 0.5% vs. 26.1 ± 6.8%, P-PMN vs. H-PMN), or decreases their 90°-SC when treated with cytochalasin B + FMLP (secretion) (2.4 ± 0.1% vs. -35.8 ± 4.6% change in 90°-SC, P-PMN vs. H-PMN), while responding comparably to C5a, PMA, and A23187. P-PMN failed to depolarize in response to FMLP but responded similarly to H-PMN when activated by C5a, A23287, and PMA. P-PMN's chemotactic response to FMLP was selectively absent since the cells responded well to purified pig C5a. FMLP stimulated significant increases in H-PMN adherence to bovine serum albumin-coated plastic (44.1 ± 6.7% vs. 12.6 ± 3.7%, FMLP vs. buffer, P < 0.025), but failed to increase adherence of P-PMN above baseline 0.68 ± 0.20% vs. 2.12 ± 1.90%, FMLP vs. buffer, P > 0.05, PMA (100 ng/ml) stimulated comparable increses in adherence in both PMN types (48.6 ± 5.2% vs. 58.7 ± 4.9%, P-PMN vs. H-PMN, P <).025). Binding studies using the flouresceinated N-formyl peptide **f-met-leu**-phe-lysine-fluorescein-isothiocyanate (FMLPL-FITC) in the absence and presence of excess non-fluoresceinated FMLP indicated that P-PMN lack specific binding sites for the **N-formyl peptides**. Intracoronary (LAD) infusion of FMLP in the instrumented intact pig produced no change in neutrophil extraction, LAD-regional blood flow or myocardial contractility while infusion of purified porcine C5a induced a rapid and marked increase in myocardial neutrophil extraction, a decrease in LAD coronary blood flow, and diminished contractility. It is concluded that P-PMN and H-PMN respond comparably to C5a, PMA, and A23187, but P-PMN are selectively unresponsive to activation by FMLP both in vitro and in vivo due to the absence of FMLP binding.

ACCESSION NUMBER: 1990:262135 BIOSIS
DOCUMENT NUMBER: PREV199090004221; BA90:4221
TITLE: IN-VIVO AND IN-VITRO ASSESSMENT OF PORCINE NEUTROPHIL ACTIVATION RESPONSES TO CHEMOATTRACTANTS FLOW CYTOMETRIC EVIDENCE FOR THE SELECTIVE ABSENCE OF FORMYL PEPTIDE RECEPTORS.
AUTHOR(S): FLETCHER M P [Reprint author]; STAHL G L; LONGHURST J C
CORPORATE SOURCE: DIV RHEUMATOL/ALLERGY, TB 192, SCH MED, UNIV CALIF AT DAVIS, DAVIS, CALIF 85616, USA
SOURCE: Journal of Leukocyte Biology, (1990) Vol. 47, No. 4, pp. 355-365.
CODEN: JLBIE7. ISSN: 0741-5400.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 5 Jun 1990
Last Updated on STN: 7 Aug 1990

L9 ANSWER 14 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI STUDIES OF SIGNAL TRANSDUCTION IN THE RESPIRATORY BURST-ASSOCIATED STIMULATION OF **F-MET-LEU**-PHE-INDUCED TUBULIN TYROSINOLATION AND PHORBOL 12-MYRISTATE 13-ACETATE-INDUCED POSTTRANSLATIONAL INCORPORATION OF TYROSINE INTO MULTIPLE PROTEINS IN ACTIVATED NEUTROPHILS AND HL-60 CELLS.
AB A specific stimulation of tubulin tyrosinolation in human neutrophils (PMNs) is induced by the synthetic peptide chemoattractant N-formylmethionylleucyl-phenylalanine (fMet-Leu-Phe), and this stimulation is closely associated with activation of the NADPH oxidase-mediated respiratory burst (Nath, J., and Gallin, J. I. (1983) J. Clin. Invest. 71, 1273-1281). In contrast, along with tubulin tyrosinolation, a distinctly different respiratory burst-associated random posttranslational incorporation of tyrosine into multiple PMN proteins is observed in PMNs stimulated with the phorbol ester phorbol 12-myristate 13-acetate (PMA) or sn-1,2-dioctanoylglycerol (DAG). In studies exploring the mechanism(s) of signal transduction for these distinct neutrophil responses, we found that the fMet-Leu-Phe-indcued stimulation of tubulin tyrosinolation in PMNs and in differentiated HL-60 cells is completely blocked by pertussis toxin, while the PMA-induced random incorporation of tyrosine is not inhibited. We also found that expression of the fMet-Leu-Phe-mediated stimulation of tubulin tyrosinolation in HL-60 cells is correlated with increases in the specific activity of protein kinase C and with the acquisition of respiratory burst activity which occur during induced myeloid maturation of these cells. Furthermore, both the fMet-Leu-Phe-induced stimulation of tubulin tyrosinolation and the PMA or DAG-induced random posttranslational incorporation of tyrosine into multiple proteins in activated neutrophils, were found to be reversibly inhibited (>70%) by the protein kinase inhibitors 1-(5-isoquionolinesulfonyl)piperazine (C-I) and 1-(5-isoquionolinesulfonyl)-2-methylpiperazine (H-7), in parallel with inhibition of superoxide (O2-) generation. In related studies, we also found that fMet-Leu-Phe-stimulated O2- production is comparably inhibited by C-I and H-7, but in a highly temperature-dependent manner. Inhibition was observed only when C-I or H-7 is added to PMNs at physiologic temperature, i.e. 37° C. Interestingly, inhibition of the PMA-induced O2- generation by C-I or H-7 was not found to be similarly temperature-dependent. Considered together, these findings argue against the suggestion that there is a protein kinase C-independent pathway for activation of the respiratory burst in neutrophils stimulated with **N-formyl peptides**.
ACCESSION NUMBER: 1989:133719 BIOSIS
DOCUMENT NUMBER: PREV198987068372; BA87:68372
TITLE: STUDIES OF SIGNAL TRANSDUCTION IN THE RESPIRATORY BURST-ASSOCIATED STIMULATION OF **F-MET-LEU**-PHE-INDUCED TUBULIN TYROSINOLATION AND PHORBOL 12-MYRISTATE 13-ACETATE-INDUCED POSTTRANSLATIONAL INCORPORATION OF TYROSINE INTO MULTIPLE PROTEINS IN ACTIVATED NEUTROPHILS AND HL-60 CELLS.
AUTHOR(S): NATH J [Reprint author]; POWLEDGE A; WRIGHT D G
CORPORATE SOURCE: DEP HEMATOL, WALTER REED ARMY INST RES, WASHINGTON, DC 20307-5100, USA
SOURCE: Journal of Biological Chemistry, (1989) Vol. 264, No. 2, pp. 848-855.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 10 Mar 1989
Last Updated on STN: 10 Mar 1989

L9 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI EVIDENCE FOR THE PRESENCE OF SPECIFIC RECEPTORS FOR N FORMYL CHEMOTACTIC PEPTIDES ON HUMAN SPERMATOZOA.

AB Synthetic N-formylated peptides are potent chemoattractants for human spermatozoa in vitro. The specific structure-activity relations for eliciting a chemotactic response and the ability of the antagonist terbutoxycarbonyl-phenylalanyl-leucyl-phenylalanyl-leucyl-phenylalanine (Boc-Phe-Leu-Phe-Leu-Phe) to inhibit the chemotaxis induced by these peptides strongly suggest the presence of receptors on human spermatozoa. The following studies were performed to identify specific binding sites on human spermatozoa by using [35S]-N-formyl-methionyl-leucyl-phenylalanine ([35S]**f-Met-Leu**-Phe), a potent chemotactic peptide. Binding of the [35S]formyl-peptide to human spermatozoa was rapid (t1/2, 8 min) and reversible. Binding isotherms of the saturation experiments revealed a single class of high affinity, low capacity binding sites (equilibrium dissociation constant, 17.7 nM; maximal binding, 109 fmol/2 + 106 cells) and an average number of 60,000 receptors per cell. The biological potencies of a series of formyl peptides as chemoattractants correlated closely with their relative abilities to compete with [35S]**f-Met-Leu**-Phe for specific binding to human spermatozoa. These data fulfill the major criteria for demonstration of specific receptors for chemotactic peptides on human spermatozoa. It is likely that these receptor sites initiate the chemotactic response of human spermatozoa to **N-formyl peptides**.

ACCESSION NUMBER: 1986:459715 BIOSIS
DOCUMENT NUMBER: PREV198682116557; BA82:116557
TITLE: EVIDENCE FOR THE PRESENCE OF SPECIFIC RECEPTORS FOR N FORMYL CHEMOTACTIC PEPTIDES ON HUMAN SPERMATOZOA.
AUTHOR(S): GNESSI L [Reprint author]; FABBRI A; SILVESTRONI L; MORETTI C; FRAIDOLI F; PERT C B; ISIDORI A
CORPORATE SOURCE: V CLIN MED POLICLIN UMBERTO I, UNIV LA SAPIENZA, 00161 ROME, ITALY
SOURCE: Journal of Clinical Endocrinology and Metabolism, (1986) Vol. 63, No. 4, pp. 841-846.
CODEN: JCEMAZ. ISSN: 0021-972X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 21 Nov 1986
Last Updated on STN: 21 Nov 1986

L9 ANSWER 16 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI MODULATING INFLUENCE OF CHEMO TACTIC FACTOR INDUCED CELL ADHESIVENESS ON GRANULOCYTE FUNCTION.
AB The importance of adhesion in regulating locomotion and accumulation of polymorphonuclear leukocytes (PMN) has remained vague. The chemotaxis of human PMN resuspended in heat-inactivated plasma was maximal toward 1-10 nM N-formyl-met-leu-phe (**f-Met-Leu**-Phe), but fell below random motility toward ≥ 100 nM. This impressive decrease of motility was paralleled by increased cell adherence on Petri dishes being minimal at 1 nM and maximal at > 10 nM **f-Met-Leu**-Phe (6 ± 1 and 37 ± 2% [SE] adherent cells, respectively). Checked by phase-contrast microscopy, cells under stimualted adhesion lost the typical bipolar shape of moving PMN and became immobilized and highly flattened. PMN, preexposed to 250 nM **f-Met-Leu**-Phe and tested after washing, retained increased adhesiveness and showed extremely low random and chemotactic motility. Preexposure to 1 nM **f-Met-Leu**-Phe had no effect of chemotaxis. Supporting the concept that immobilizing hyperadhesiveness does not correspond to a general functional hyporesponsiveness of PMN, no depression of the initial ingestion rate was observed in the presence of 250 nM **f-Met-Leu**-Phe. A close correlation was found between the induction of PMN adhesiveness and the stimulation of the hexose monophosphate pathway activity as well as of lysomal enzyme release ($r \geq 0.98$). Chemotactic deactivation and high-dose inhibition of chemotaxis by

N-formyl peptides is the consequence of increased cell adhesiveness. This phenomenon provides a mechanism for cell trapping at the inflammatory site. If operative in circulating blood, e.g., in septicemia, it may impair PMN emigration to such sites.

ACCESSION NUMBER: 1979:268969 BIOSIS
DOCUMENT NUMBER: PREV197968071473; BA68:71473
TITLE: MODULATING INFLUENCE OF CHEMO TACTIC FACTOR INDUCED CELL ADHESIVENESS ON GRANULOCYTE FUNCTION.
AUTHOR(S): FEHR J [Reprint author]; DAHINDEN C
CORPORATE SOURCE: SECT HEMATOL CH5, DEP MED, UNIV ZUR, CH-8091 ZURICH, SWITZ
SOURCE: Journal of Clinical Investigation, (1979) Vol. 64, No. 1, pp. 8-16.
CODEN: JCINAO. ISSN: 0021-9738.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

L9 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Modulation of alpha-6 integrin-mediated responses
AB A method for modulating an alpha 6 subunit containing integrin-mediated signal transduction is described. The method involves contacting a cell with an effective integrin modulating amount of an alpha 6 subunit containing integrin-mediated signal transduction pathway modification agent. Preferred agents are **N-formyl peptides** having the formula **f-Met-Leu**-X, wherein X is selected from the group consisting of Tyr, Tyr-Phe, Phe-Phe and Phe-Tyr. The method can be used to treat and VLA-6 integrin-mediated pathol. conditions such as the pro-inflammatory response, cancer metastasis or coronary heart disease.

ACCESSION NUMBER: 2001:868244 HCAPLUS
DOCUMENT NUMBER: 136:626
TITLE: Modulation of alpha-6 integrin-mediated responses
INVENTOR(S): Clagett, James; Lipani, John; Palmer, Craig
PATENT ASSIGNEE(S): Histatek, LLC, USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001089552	A1	20011129	WO 2001-US16774	20010523
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1283715	A1	20030219	EP 2001-939365	20010523
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003050249	A1	20030313	US 2001-863837	20010523
BR 2001011083	A	20030408	BR 2001-11083	20010523
JP 2003534288	T2	20031118	JP 2001-585795	20010523

PRIORITY APPLN. INFO.: US 2000-206397P P 20000523
WO 2001-US16774 W 20010523

OTHER SOURCE(S): MARPAT 136:626
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 18 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: flow cytometric evidence for the selective absence of formyl peptide receptors
AB Interest in the role that activated granulocytes play in complement C5a-induced myocardial ischemia prompted an investigation and comparison of the activation responses of pig and human neutrophils. The responses of Hypaque-Ficoll purified porcine (P-PMN) and human neutrophils (H-PMN) to stimulation with N-formyl-Met-Leu-Phe (FMLP), C5a, phorbol myristate acetate (PMA), and Ca ionophore A23187 were compared by flow cytometrically measured changes in the cells' forward (FWD-SC) (a measure of shape/volume change) and right angle (90°-SC) light scatter (a measure of secretion), and in the distribution of the membrane potential sensitive fluorescent probe di-O-C(5) (3). FMLP, C5a, and zymosan-activated serum (ZAS) stimulated chemotaxis and FMLP vs. PMA-stimulated adherence to plastic were also compared. Unstimulated P-PMN had lower FWD-SC and 90°-SC than H-PMN. P-PMN selectively failed to increase their FWD-SC upon stimulation with FMLP or decrease their 90°-SC when treated with cytochalasin B + FMLP (secretion), while responding comparably to C5a, PMA, and A23187. P-PMN failed to depolarize in response to FMLP but responded similarly to H-PMN when activated by C5a, A23187, and PMA. P-PMN's chemotactic response to FMLP was selectively absent since the cells responded well to purified pig C5a. FMLP stimulated H-PMN adherence to bovine serum albumin-coated plastic, but failed to increase adherence of P-PMN above baseline stimulated comparable increases in adherence in both PMN types. Binding studies using the fluoresceinated N-formyl peptide **f-Met-Leu**-Phe-Lys-fluorescein-isothiocyanate (FMLPL-FITC) in the absence and presence of excess non-fluoresceinated FMLPL indicated that P-PMN lack specific binding sites for the **N-formyl peptides**. Intracoronary (LAD) infusion of FMLP in the instrumented intact pig produced no change in neutrophil extraction, LAD regional blood flow, or myocardial contractility while infusion of purified porcine C5a induced a rapid and marked increase in myocardial neutrophil extraction, a decrease in LAD coronary blood flow, and diminished contractility. Thus, P-PMN and H-PMN respond comparably to C5a, PMA, and A23187, but P-PMN are selectively unresponsive to activation by FMLP both in vitro and in vivo due to the absence of FMLP binding.

ACCESSION NUMBER:	1990:233437 HCAPLUS
DOCUMENT NUMBER:	112:233437
TITLE:	In vivo and in vitro assessment of porcine neutrophil activation responses to chemoattractants: flow cytometric evidence for the selective absence of formyl peptide receptors
AUTHOR(S):	Fletcher, Mark P.; Stahl, Gregory L.; Longhurst, John C.
CORPORATE SOURCE:	Sch. Med., Univ. California, Davis, CA, 95616, USA
SOURCE:	Journal of Leukocyte Biology (1990), 47(4), 355-65 CODEN: JLBIE7; ISSN: 0741-5400
DOCUMENT TYPE:	Journal
LANGUAGE:	English

L9 ANSWER 19 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Modulating influence of chemotactic factor-induced cell adhesiveness on granulocyte function
AB Chemotaxis of human polymorphonuclear leukocytes (PMN) resuspended in heat-inactivated plasma was maximal toward 1-10 nM N-formyl-met-leu-phe (**f-Met-Leu**-Phe), but fell below random motility toward ≥100 nM. This decrease of motility was paralleled by increased cell adherence, being minimal at 1 nM and maximal at >10 nM **f-Met-Leu**-Phe (6 and 37% adherent cells, resp.). Cells under stimulated adhesion lost the typical bipolar shape of moving PMN and became immobilized and highly flattened. PMN, preexposed

to 250 nM **f-Met-Leu**-Phe and tested after
washing, retained increased adhesiveness and showed extremely low random
and chemotactic motility. In contrast, preexposure to 1 nM **f-Met-Leu**-Phe had no effect on chemotaxis. Supporting the
concept that immobilizing hyperadhesiveness does not correspond to a
general functional hyporesponsiveness of PMN, no depression of the initial
ingestion rate was observed in the presence of 250 nM **f-Met-Leu**-Phe. Moreover, a close correlation was found between the
induction of PMN adhesiveness and the stimulation of the hexose
monophosphate pathway activity as well as of lysosomal enzyme release.
Thus, chemotactic deactivation and high-dose inhibition of chemotaxis by
N-formyl peptides is the consequence of
increased cell adhesiveness. This phenomenon provides a mechanism for
cell trapping at the inflammatory site. Conversely, if operative in
circulating blood, e.g., in septicemia, it may impair PMN emigration to
such sites.

ACCESSION NUMBER: 1979:506484 HCAPLUS
DOCUMENT NUMBER: 91:106484
TITLE: Modulating influence of chemotactic factor-induced cell adhesiveness on granulocyte function
AUTHOR(S): Fehr, Jorg; Dahinden, Clemens
CORPORATE SOURCE: Dep. Med., Univ. Zurich, Zurich, CH-8091, Switz.
SOURCE: Journal of Clinical Investigation (1979), 64(1), 8-16
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English

=> d his

(FILE 'HOME' ENTERED AT 16:15:06 ON 04 FEB 2004)

FILE 'STNGUIDE' ENTERED AT 16:15:13 ON 04 FEB 2004

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JAPIO, BIOSIS, CEN, CEABA-VTB, BIOBUSINESS, HCAPLUS' ENTERED AT 16:16:22 ON 04 FEB 2004

L1 993 S FIBROSIS () TREATMENT
L2 45818 S FIBROSIS AND CIRRHOSIS
L3 1 S CHRONIC PANCREATITUS
L4 95 S L2 AND L1
L5 2524 S F-MET-LEU
L6 346 S N-FORMYL PEPTIDES
L7 0 S L6 AND L1
L8 1 S L5 AND L1
L9 19 S L5 AND L6

=> fibrosis
FIBROSIS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s fibrosis
L10 365786 FIBROSIS

=> s l10 and vas deferens repair
L11 0 L10 AND VAS DEFERENS REPAIR

=> s l10 and fallopian tube repair
L12 1 L10 AND FALLOPIAN TUBE REPAIR

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L12 ANSWER 1 OF 1 USPATFULL on STN

TI 207 human secreted proteins

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:258639 USPATFULL

TITLE: 207 human secreted proteins

INVENTOR(S): Ni, Jian, Germantown, MD, UNITED STATES
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	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003181692	A1	20030925
APPLICATION INFO.:	US 2001-933767	A1	20010822 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2001-US5614, filed on 21 Feb 2001, PENDING Continuation-in-part of Ser. No. US 1998-205258, filed on 4 Dec 1998, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-184836P	20000224 (60)
	US 2000-193170P	20000329 (60)
	US 1997-48885P	19970606 (60)
	US 1997-49375P	19970606 (60)
	US 1997-48881P	19970606 (60)
	US 1997-48880P	19970606 (60)
	US 1997-48896P	19970606 (60)
	US 1997-49020P	19970606 (60)
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US 1997-57634P 19970905 (60)
US 1997-70923P 19971218 (60)
US 1998-92921P 19980715 (60)
US 1998-94657P 19980730 (60)
US 1997-70923P 19971218 (60)